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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
CHUNDURU, SURYAPRABHA	
ART UNIT	**PAPER NUMBER**
1637	

DATE MAILED: 08/08/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/021,955	LUPSKI ET AL.	
	Examiner	Art Unit	
	Suryaprabha Chunduru	1637	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *30 May 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-7,35-40 and 43-61* is/are pending in the application.

4a) Of the above claim(s) *6,37,42 and 47* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-5, 7, 35-36, 38-40, 43-46, 48-61* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

1. Upon Vacatur of the case by the BPAI, the case is reopened to address the issues raised by the BPAI.

Status of the application

2. Claims 1-7, 35-40, 42-61 are pending. The original claims 1-40 were subjected to a restriction requirement. The claims were further restricted for species and SEQ ID No. election.

Applicants' elected Group I (claims 1-13) along with the species (274Δ) and SEQ ID No. 76. with traverse and requested rejoinder of Groups I and IX . Examiner rejoined Groups I and IX (claims 1-13, 35-40). Claims 41-61 are added subsequently. Claims 6, 37, 42, 47 read on non-elected SEQ ID No. and species and are withdrawn from further consideration. Claims 1-5, 7, 35-36, 38-40, 43-46, 48-61 are considered for examination along with the elected SEQ ID No. 76 and species 274Δ. Claims 2-3, 38, 44-45, 50 read on elected SEQ ID No. 76. Claims 7, 36,48 read on the elected species 274ΔC. According to MPEP 8-09, Claims 1, 4-5,35, 39, 40, 43, 46, 49, 51-61 link(s) inventions I and IX. According to MPEP 8-12 the generic or other linking claims should not be associated with any one of the linked inventions since such claims must be examined with the elected invention. See MPEP § 809. Thus claims 1, 4-5,35, 39, 40, 43, 46, 49, 51-61are considered as linking claims that are examined with the elected species and elected SEQ ID No. 76.

Response to BPAI decision

3. The Board raised the issue that the rejection of claims 1-7, 35-40, 42-51 under 35 USC 112, first paragraph (lack of enablement) is based on broad claims and the rejection briefly discuss the

elected SEQ ID No. and the species 274ΔC. To address this issue examiner briefly describe the linking claims as follows.

The broad claims 1, 4-5, 35, 39, 40, 43, 46, 49, 51-61 are linking claims and have been examined . Examination of such claims was not limited to either the species or sequence election. Claims 2-3, 38, 44-45 and 50 have been examined as they read on elected SEQ ID No. 76. Claims 7, 36, and 48 have been examined as they read on the elected species 274Δ. Thus considering linking claim practice, Examiner has addressed the lack of enablement of these broad claims and their association with the elected SEQ ID No. 76, species 274ΔC. The non-elected SEQ ID Nos. 1, 27-75, and 77 are genus of periaxin polynucleotides each comprising mutations or polymorphisms. The non-elected SEQ ID Nos. 3-26 are genus of primers for amplifying periaxin polynucleotides.

The Board also raised the issue that the claims 35 and 49 do not require any diagnosis and advises the examiner to consider the claims separately in making a lack of enablement rejection. Examiner notes that the claims 35 and 49 are drawn to a method for detecting the presence or absence of a mutation or polymorphism associated with a myelinopathy, said myelinopathy resulting from a periaxin mutation in an individual. It is noted that the claims 35 and 49 are not a mere detection assay but they represent the association of said periaxin mutation with a myelinopathy, thus the claims are drawn to a detection of a periaxin mutation and diagnosis of a myelinopathy. Therefore the rejection does apply to the claims 35 and 49.

Considering the above issues Examiner reopens the prosecution to address the rejection under 25 USC 112, first paragraph (enablement) with regard to elected claims as well as adding new grounds of rejections under 35 Usc 112 first paragraph written description.

Claim Rejections - 35 USC § 112

4. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1-5, 7, 35-36, 38-40 and 43-46, 48-61 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

There are many factors to be considered when determining whether there is sufficient evidence to support determination that a disclosure does not satisfy the enablement requirements and whether any necessary experimentation is undue (see In re Wands, 858 F. 2d 731, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). These factors include, but are not limited to:

Quantity of Experimentation Necessary

Amount of Direction and Guidance

Presence and Absence of Working Examples

Nature of Invention

Level of Predictability and unpredictability in the art

Nature of the Invention :

Claims 1-5, 7 are drawn to a method of diagnosing myelinopathy in an individual and claims 35, 49 are drawn to a method of detecting the presence or absence of any mutation in a periaxin polynucleotide and its association with a myelinopathy. It is noted that the claims 35 and 49 are not a mere detection assay since they require an association of said periaxin mutation with a myelinopathy. Thus the method implies diagnosis based detection of a periaxin mutation in myelinopathy. Claim 57 and 61 are drawn to a method for identifying an individual suspected of having myelinopathy or being a carrier of myelinopathy. Further, Claims 2, 38, 44-45, 50 are drawn to a method for diagnosing / detecting periaxin polynucleotide comprising elected SEQ ID No. 76. Claims 7, 36, 48 are drawn to elected species 274ΔC, an alteration in a periaxin polynucleotide and its association with a myelinopathy. Claims 4 and 40 are broadly drawn to a broad range of diseases of myelinopathy such as Charcot-Marie-Tooth (CMT) syndrome, hereditary neuropathy with liability to pressure palsies (HNPP), Dejerine-Sottas syndrome (DSS), congenital hypomyelinating neuropathy (CHN), and Roussy-Levy syndrome (RLS). Claim 50 is drawn to a DSS. Claim 58 drawn to a prominent sensory neuropathy. Claims 53-56 are drawn to a method of identifying alteration comprising homozygous, heterozygous and compound heterozygous periaxin mutations.

Amount of Direction and Guidance:

The specification discloses the identity of several mutations in periaxin polynucleotide and their locations (see Figs. 4 and 9). The specification on page 14, asserts a correlation between the human orthologue of murine and rat periaxin (PRX) with human inherited myelinopathy and further asserts that human periaxin gene which encodes two PDZ domain proteins, is required for the maintenance of peripheral nerve myelin. The specification teaches that based on knockout

animal models, periaxin is correlated to the proper formation of myelin sheaths and the specification broadly discloses the identification of recessive PRX mutations comprising nonsense and frame shift mutations in the periaxin gene. The specification asserts that based on the common known methods in the art, mutations in other periaxin polynucleotide sequences (for example SEQ ID No. 76) could be detected. The specification discloses mutations in SEQ ID No.1 and extrapolates the use of similar techniques to detect mutations in other periaxin polynucleotides (for example SEQ ID NO.76). The specification discloses mutations in other genes associated with some myelinopathy (see page 20) (such as DNA rearrangements in CMT patients caused by mutations in MPZ, Cx32, EGR2, and mutations in MPZ and EGR2 in DSS patients). Further the specification on page 21, asserts the function of periaxin in the maintenance of the myelin sheath based on animal studies. However, the specification has not established that a statistically significant association exists between all of the specific mutations disclosed in the specification, and any myelinopathy, or any specific myelinopathy, or that a predictable correlation can be made as to an association between any mutation in the periaxin gene and any myelinopathy or any specific myelinopathy. Further the specification has not established that any periaxin mutation is associated with DSN and no predictable correlation is established that any homozygous periaxin mutation or that two different mutations in a compound heterozygote are associated with myelinopathy in general. With regard to the new claims 51-61, the specification (on page 65, example 3) provides evidence for compound heterozygous with a deletion and a transition mutation associated with specific types of myelinopathies that is one form of CMT and DSN. However, the specification fails to establish that any alteration in PRX is diagnostic for myelinopathy in general, nor that the presence of a single mutation in a single allele would

indicate that someone is susceptible to myelinopathy or a carrier of a periaxin associated myelinopathy as it is clear that mutations in PRX exist which are not only not diagnostic but also not associated with myelinopathy (table 2). The specification exemplifies that the presence of an alteration in the periaxin gene is not necessarily diagnostic for myelinopathies or an indicator that someone is a carrier for a disease causing mutation, as broadly claimed.

Presence and Absence of working examples:

The specification discloses a method of screening Prx mutations in some family studies and detected mutations comprising a deletion and a transition in the affected patients with peripheral neuropathy. The specification correlates the mutations with the loss of function of PRX gene in relation to studies in rat (example 4). The examples 2-4 in the specification establish a positive correlation between the presence of a periaxin polynucleotide comprising mutation which results in a truncated periaxin polypeptide in patients with undisclosed myelinopathy, wherein said patients have two aberrant forms of periaxin polypeptides. Although the specification does not demonstrate any alteration in PRX is associated with myelinopathies in general, the specification asserts that the mutations could be associated with loss of function of the periaxin polypeptide. It is noted, however, that these examples also establish that the mere presence of a mutation (i.e., only a single copy) is NOT associated with the disease as in HOU579 family, wherein the unaffected parents had an allele with single mutated PRX polynucleotide and another allele of wild type PRX polynucleotide. Further there is no description of the type of peripheral neuropathy of the affected patients. (see page 65, example 3). Further examples in the specification merely asserts correlation between mutations in PRX with myelinopathy in general, however no specific mutation is associated with any of the different types of myelinopathies as

exemplified by the example 8 in the specification (see page 72). Further Table- 2 shows that the unaffected control subjects contain mutations in periaxin. The specification does not teach whether the mutations in Table-2 are associated with loss of function or if they are statistically associated with any specific peripheral neuropathy or any specific myelinopathy. Thus the mere detection of an alteration in PRX gene is not diagnostic for myelinopathies in general. The specification fails to show that all alterations are diagnostic or associated with myelinopathies because the specification shows that carriers having an alteration are unaffected with myelinopathy (Table-2). Thus all carriers having an alteration or mutation in PRX would not be carriers of disease-causing PRX mutations. The specification does not provide guidance as to which PRX alterations are predictably associated with myelinopathy in general, or not associated with myelinopathy. Further the specification does not provide guidance as to which PRX alterations would indicate an individual being a carrier of disease-causing mutations or not.

Level of Predictability and unpredictability in the art :

Predictability in the art suggests mutations in genes other than the specific periaxin gene, are associated with specific type of myelinopathy, for example Boss et al. (USPN. 5,691,144) teaches mutations in connexin-32 are associated with X-linked Charcot-Marie-Tooth (CMT) disease, Timmermann et al. (Neurology, Vol. 52, pp. 1827-1832, 1999) teach a missense mutation in EGR2 gene in association with Dejerine-Scottas syndrome (DSS). Lupski et al. (USPN. 5,780,223) teach DNA duplication in CMT1A gene sequence association with autosomal dominant CMT disease, and Roa et al. (Nature Genetics, Vol. 5, pp. 269273, 1993) teach that some point mutations in peripheral myelin protein 22 (PMP22) gene are associated with CMT1A, while others are associated with DSS (Fig.3, page 271). With regards to the

specific periaxin gene Guilbot et al. (Human Molecular Genetics, Vol. 10, No.4, 2001), teach periaxin is responsible for CMT4F, an autosomal recessive form of CMT disease, and Gillepsie et al. (Neuron, Vol. 12, pp. 497-508, 1994) teach role of periaxin in rat peripheral nervous system and discloses that periaxin localization in schwann cells and its possible role in ensheathment. Further, Takashima et al. disclose periaxin mutations cause a broad spectrum of demyelinating neuropathies and disclose that affected patients with CMT or DSN comprise PRX mutations in homozygous condition, that is both alleles are mutated with a specific mutation and the unaffected family members are carriers for myelinopathy, that is a single allele of PRX is mutated (Takashima et al. Ann. Neurol., Vol. 51, pp. 709-715, 2002). Kijima et al. disclose yet another Prx mutation causing early-onset but slow-progressive CMT disease (Kijima et al. J Hum Genet., Vol. 49, pp. 376-379, 2004). However, the art does not establish a predictable association that any specific mutation in periaxin or any other genes is predictably associated with all of the large number of diseases encompassed by the recitation of "myelinopathy". For example Roa et al. teach that while some point mutations in PMP22 are associated with CMT1A, others are associated with DSS. Further, while Boerkoel et al. (Am. J. Hum. Genet., Vol. 68, pages 325-333, 2001) teach that certain specific mutations are associated with DSN, when both copies of periaxin gene are altered, Boerkoel et al. further teach that the family members with only one altered copy of periaxin gene were not affected and also teach a number of missense mutations in normal and unaffected family members. Further, Takashima et al. teach similar study, wherein DSN or CMT affected patients have two mutated alleles where as the unaffected have one mutated allele with no demyelination. The art is further silent with regard to a predictable association between any specific alteration or mutation in periaxin and a

representative number of diseases encompassed by the term "myelinopathy". Diseases encompassed by the term "myelinopathy" include a large number of heterogeneous diseases with differing symptoms and associations to genetic mutations. To date, however, there is no evidence that the association of an alteration or mutation in a specific gene and a specific form of myelinopathy can predictably correlate the presence of any other, or all, specific myelinopathy encompassed by the broad term "myelinopathy". The claims further broadly encompass detecting an association between any specific mutation in periaxin, and an association to a specific unnamed myelinopathy. The specification, however does not establish a statistically significant association with any of the disclosed mutations in periaxin, and any specific form of myelinopathy such that the skilled artisan might be able to predictably correlate which mutations in periaxin would be associated with a specific form of myelinopathy. The mere detection of an alteration in PRX gene is not associated with myelinopathies in general. Further, to date, no teaching is available in the art with regards to a universal correlation between any mutation in periaxin and an association with any general or specific type of myelinopathy. It is apparent from the prior art that the unpredictability is high and the instant specification fails to teach any particular mutation associated with any particular type of myelinopathy. Given the broad scope of the claims, the specification does not provide any specific example that would easily predict a significant association of any particular mutation in periaxin with any particular type of myelinopathy. Further, CMT is inherited in three forms, i.e., autosomal dominant, autosomal recessive and X-linked conditions. The specification fails to support an association of a mutation in periaxin with all the three forms of CMT.

In addition, the specification does not establish the identity of any specific critical nucleotide or amino acid alteration(s) that are associated with loss of function or are associated with myelinopathy. The missense mutations in Table-2 were also found in unaffected controls. The specification does not teach if patients had one copy of mutation, or if they were homozygous or compound heterozygous for other mutations in periaxin. With regard to the data in Table 2, examiner notes that table-2 shows PRX mutations in unaffected controls. The specification does not teach if patients had one copy of mutation, or if they were homozygous or compound heterozygous for other mutations in periaxin. It is clear from the teachings in Table-2, that the mere detection of the presence of an alteration in periaxin such as a substitution or deletion is not diagnostic for myelinopathy in general. Further, with regard to the 2145T-> A and 274Δ C mutation in claim 36, and the R196X, C715X, or R82FSX96, the specification has provided no data as to whether these mutations are even associated with myelinopathy. Since the specification has not identified any specific critical amino acid alteration, it is further unclear whether these mutations or any specific mutation will have a significant affect or not. With regard to the heterozygous carriers carrying a PRX mutant allele, the specification fails to establish that the mere presence of an alteration in PRX as claimed broadly in the new claims 51-61 would result in carriers of disease causing PRX mutant alleles. The claims are drawn to methods of diagnosing or determining increased susceptibility or a carrier status merely based on detection of an alteration. However, as exemplified by the specification all alterations in PRX are not predictably correlated with myelinopathies, including prominent sensory neuropathies. With regard to the heterozygous carriers carrying a PRX mutant allele, the specification fails to establish that the mere presence of an alteration in PRX as claimed broadly in the new claims 51-

61 would result in carriers of disease causing PRX mutant alleles, or result in susceptibility to any myelinopathy.

Quantity of Experimentation Necessary:

Given the lack of guidance in the specification and the unpredictability in the art, it would require a large amount of experimentation to practice the invention as claimed. Neither the art nor the specification provides the skilled artisan with a predictable correlation that any mutation in periaxin is significantly associated with any specific myelinopathy. To practice the invention as claimed, the skilled artisan would have to perform a large study of patients with different types of myelinopathy, such as CMT, DSS, and matched controls to determine if any general alteration or mutation in periaxin or any specific claimed alteration or mutation in peraxin, was associated with any myelinopathy in general. Such a study would consist of mainly trial and error analysis, the outcome of which is clearly unpredictable as exemplified by the state of the art. Further, it would require a large amount of experimentation to distinguish, which PRX mutant allele carriers are associated with the myelinopathy in general or which PRX mutant allele carriers are not associated with myelinopathy. Thus a mere presence of an alteration is not diagnostic for any myelinopathy or is not diagnostic for identifying a carrier having a disease-causing PRX mutant allele. Therefore, given the lack of guidance from the specification as to any statistical association between the claimed association of any mutation in peraxin polynucleotide and any myelinopathy, and the unpredictability taught in the art as to some point mutations in other genes such as PMP22 are associated with one form of CMT, while other mutations in the same PMP22 are associated with DSS, the skilled artisan would be required to perform undue experimentation to practice the invention as broadly as it is claimed.

Claim Rejections - 35 USC § 112

5. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1, 4-5, 35, 39, 40, 43, 46, 49, 51-61 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

In analysis of the claims for compliance with the written description requirement of 35 U.S.C. 112, first paragraph, the written description guidelines note regarding genus/species situations that "Satisfactory disclosure of a ``representative number" depends on whether one of skill in the art would recognize that the applicant was in possession of the necessary common attributes or features of the elements possessed by the members of the genus in view of the species disclosed." (See: Federal Register: December 21, 1999 (Volume 64, Number 244), revised guidelines for written description.)

All of the current claims encompass a genus of periaxin nucleic acids which comprise a large number of mutations or polymorphisms which are not disclosed in the specification. The genus includes an enormous number of periaxin sequences or fragments comprising mutations or polymorphisms for which no written description is provided in the specification. The specification only disclosed a few of the particularly named polymorphisms and mutations set forth in represented in table-2 of the specification on page 67. The table-2 shows alterations in

unaffected family members (controls), that are not associated with any particular phenotype, on contrary in Fig. 4 and 10 the association of some specific alterations were shown demonstrating an association with the DSN or CMT phenotype. Thus, applicant has expressed possession of only a few of particular polymorphisms, in a genus, which comprises hundreds of millions of different possibilities, that are associated with a particular phenotype.. Here, no common element or attributes of the sequences are disclosed which would permit selection of sequences as polymorphisms. Thus the specification disclosed that some mutations are associated with a specific myelinopathy and some are not associated with any myelinopathy. There is no disclosed functional correlation between the structure of the mutations and either their function in myelinopathy or how they affect the function of priaxin protein.. The species disclosed are not representation of the large genus of possible polymorphisms or mutations associated with specific myelinopathy. Further, these claims expressly encompass all the different possible allelic variants including insertions, deletion, substitutions and transversions at thousands of different sites. No written description of alleles, of upstream or downstream regions containing additional sequence, which are associated with any phenotype are described in the specification.

It is noted in the recently decided case The Regents of the University of California v. Eli Lilly and Co. 43 USPQ2d 1398 (Fed. Cir. 1997) decision by the CAFC that "A definition by function, as we have previously indicated, does not suffice to define the genus because it is only an indication of what the gene does, rather than what it is. See Fiers, 984 F.2d at 1169- 71, 25 USPQ2d at 1605- 06 (discussing Amgen). It is only a definition of a useful result rather than a definition of what achieves that result. Many such genes may achieve that result. The description requirement of the patent statute requires a description of an invention, not an indication of a

result that one might achieve if one made that invention. See In re Wilder, 736 F.2d 1516, 1521, 222 USPQ 369, 372- 73 (Fed. Cir. 1984) (affirming rejection because the specification does "little more than outlin[e] goals appellants hope the claimed invention achieves and the problems the invention will hopefully ameliorate."). Accordingly, naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material.

> It is noted that in Fiers v. Sugano (25 USPQ2d, 1601), the Fed. Cir. concluded that "...if inventor is unable to envision detailed chemical structure of DNA sequence coding for specific protein, as well as method of obtaining it, then conception is not achieved until reduction to practice has occurred, that is, until after gene has been isolated...conception of any chemical substance, requires definition of that substance other than by its functional utility."

In the instant application, certain specific SEQ ID NOs are described. Also, in Vas-Cath Inc. v. Mahurkar (19 USPQ2d 1111, CAFC 1991), it was concluded that:

> "...applicant must also convey, with reasonable clarity to those skilled in art, that applicant, as of filing date sought, was in possession of invention, with invention being, for purposes of "written description" inquiry, whatever is presently claimed."

In the application at the time of filing, there is no record or description which would demonstrate conception of any nucleic acids other than those expressly disclosed which comprise a mutation or a polymorphism. Therefore, the claims fail to meet the written description requirement by encompassing sequences which are not described in the specification.

Conclusion

No claims are allowable.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Suryaprabha Chunduru whose telephone number is 571-272-0783. The examiner can normally be reached on 8.30A.M. - 4.30P.M , Mon - Friday,.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Benzion can be reached on 571-272-0782. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

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